increased antioxidant activity], the plain β-blocking agent Carazolol [CAR] with a carbazol moiety (like C), the α-blockers Prazosin [PRA] or Doxazosin [DOX], or the antioxidants vitamin C (VIT C, ascorbic acid) or vitamin E (VIT E, RRR-α-tocopherol). The growth factor PDGF was added as a stimulant for proliferation (DNA synthesis was assessed by ^{3}H-thymidine uptake). The effect of VIT E was measured by using a new technique for cellular VIT E loading.

Results:

Agent	Inhibition of SMC proliferation (IC_{50})
C	1 μmol
BM	1 μmol
PRA	1–10 μmol
DOX	1–10 μmol
VIT C	no inhibition
VIT E	no inhibition
CAR	no inhibition

Conclusions: SMC proliferation was inhibited by C but not by CAR. Our data show that the inhibition of aortal SMC proliferation by C is probably due to its α-blocking effect since the tested compounds without α-blocking activity revealed no inhibitory effect. Surprisingly and in contrast to the literature, the antioxidant VIT E did not show an inhibition of SMCs cultured in vitro.

1199 Cardiac Transplantation: Predictors of Outcome II

Wednesday, April 1, 1998, Noon–2:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 1:00 p.m.–2:00 p.m.

1199-53 Partial Left Ventriculectomy in Patients With end Stage Heart Disease – Initial Experience

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Purpose: To evaluate the local experience of partial left ventriculectomy (PLV) for the treatment of patients with end stage heart disease (ESHD).

Methods: 27 ESHD patients (18 males, 9 females; mean ± SD age 62 ± 13 years) underwent PLV in the period 9/96–7/97. All 27 patients had cardiomegaly and 17 patients were ineligible for heart transplantation due to medical contraindications. Idiopathic cardiomyopathy was diagnosed in 23 patients and ischemic heart disease was present in 13 patients. Left ventricular end-diastolic diameter (LVED) was 7.5 ± 0.9 cm, and ejection fraction was 15 ± 6% prior to surgery. Mitral regurgitation was present in 26 patients, and severe tricuspid regurgitation was documented in 1 individual. Concurrent operations performed with PLV included mitral valve repair in 10 patients, mitral valve replacement in 16 patients, coronary bypass in 9 patients, and tricuspid valve repair in 1 patient. During PLV, the resected ventricular mass was 55 ± 23 grams with mean dimensions of 9.1 cm × 5.6 cm.

Results: Postoperatively, ejection fraction increased by 21 ± 10%, and LVED decreased by 2.3 ± 1.2 cm. Ventricular rupture was not observed. Intravenous inotropes were required temporarily in all patients due to low-output syndrome. Reversible ventricular tachyarrhythmias occurred in 3 patients. Inducible ventricular arrhythmia was demonstrated in electrophysiologic studies in 2 patients. Transient renal insufficiency was common, though only 1 individual required dialysis after surgery. Six deaths occurred within 30 days postoperatively and 4 other patients expired after the first 30 days. Eight of the 10 deaths occurred in patients who were medically ineligible for heart transplantation prior to PLV. The remaining 17 patients (63%) remained in NYHA functional class II.

Conclusion: Satisfactory outcome was achievable with PLV in ESHD patients with dilated cardiomyopathy, including those with concomitant valvular and coronary artery lesions.

1199-54 The Predictive Impact of Abnormalities in Ventricular Repolarization on Noncellular Allograft Rejection With Hemodynamic Compromise

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Background: Whether alterations in ventricular repolarization accompany hemodynamically significant noncellular (humoral) cardiac allograft rejection remains unknown.

Methods: We investigated serial changes in ventricular repolarization abnormalities by measuring QT dispersion (QT-d) pre, during, and following recovery from acute allograft dysfunction in 15 heart transplant recipients (age 49 ± 11; 7 men) with acute hemodynamic collapse (CI < 2.21/min/m^2), evidence of complement and immunoglobulin deposition, and absence of cellular rejection on endomyocardial biopsy. QT-d intervals were measured in at least 8 of 12 leads of surface EKG obtained prior to, during, and following recovery after treatment with plasmapheresis. Rate corrected QTc-d was calculated by subtracting the shortest QT-interval from the longest interval and application of Bazett's formula.

Results: QTc-d during rejection increased significantly from baseline (76 ± 22 vs 58 ± 12; p < 0.01). Similarly, QTc-d returned to baseline levels following hemodynamic recovery (53 ± 9; p < 0.01).

	% Δ QTcd	% Δ CI	R Value
Rejection[a]	+31*	−49*	0.7*
Recovery[@]	−29*	+4.0*	0.6*

*p < 0.01; [a]compared to baseline; [@]compared to rejection

Conclusions: Alterations in indices of ventricular repolarization accompany acute allograft rejection with hemodynamic compromise. The close association of worsening repolarization during rejection and normalization of these indices with hemodynamic recovery suggest that assessment of repolarization dispersion may serve as a sensitive non-invasive marker of resolution of allograft dysfunction.

1199-55 Effect of Angiographic Abnormalities on Myocardial Perfusion and Resistance in the Cardiac Allograft

T.J. Donohue, T.L. Wolford, R.G. Bach, E.A. Caracciolo, M.J. Kern, L.A. Miller, D.S. Yip. *Saint Louis University, USA*

Pathology studies have demonstrated that transplant arteriopathy involves both epicardial and intramyocardial vessels. Coronary resistance (CR) is inversely related to microvascular cross-sectional area and is expected to be increased in patients with any angiographic abnormalities. To assess the effect of mild angiographic abnormalities on myocardial perfusion and CR post-transplant, we studied 86 allograft recipients with normal left ventricular function and mild (≤40% stenosis; abn) or no angiographic abnormalities (norm). Resting (Rest) and hyperemic (HYP) coronary blood flow was measured in the LAD, CFX, and RCA (Doppler guidewire and adenosine). CR (dynes.sec.cm^{-5}) and myocardial perfusion (mL/gm/min) were calculated and normalized for left ventricular mass. Myocardial perfusion reserve (MPR) = HYP perfusion/resting perfusion.

N	Rest Perf	HYP Perf	REST CR	HYP CR	MPR
Norm (49)	1.2 ± 0.5	3.6 ± 1	217 ± 110	63 ± 23	3.3 ± 0.8
ABN (37)	0.9 ± 0.3	3.0 ± 1	287 ± 111*	88 ± 29*	3.3 ± 0.8

*p < 0.01 v. norm

CR was significantly elevated in the abn group while perfusion was higher in the norm group. MPR, however was not different between groups, and thus the degree of microvascular vasodilatory capacity was identical. This suggests no loss of microvascular function despite the presence of conduit vessel disease in the cardiac allograft.

1199-56 Effect of Time Post-transplant on Myocardial Perfusion and Coronary Resistance

T.J. Donohue, T.L. Wolford, L.A. Miller, R.G. Bach, E.A. Caracciolo, D.S. Yip. *Saint Louis University, USA*

A reduced coronary flow reserve in the early post-transplant period has been described. This was presumed secondary to an early reduction in microvascular vasodilatory capacity and an increase in coronary resistance (CR). To assess the effect of time post-transplant on perfusion and CR we studied 86 cardiac allograft recipients with normal ventricular function and mild (<40% diameter stenosis) or no angiographic abnormalities. Patients studied ≤40 days post-transplant (early) were compared to those studied ≥1 year post-transplant (late). Resting (Rest) and hyperemic (HYP) coronary blood flow was measured in the LAD, CFX, and RCA (Doppler guidewire and adenosine). Myocardial perfusion (mL/gm/min) and coronary resistance (dynes.sec.cm^{-5}) were calculated and normalized for left ventricular mass at Rest and peak HYP. Myocardial perfusion reserve (MPR) = HYP perfusion/Rest perfusion.

N	Rest Perf.	HYP Perf	Rest CR	HYP CR	MPR
Early (19)	1.3 ± 0.4	3.7 ± 1	181 ± 77	63 ± 19	2.8 ± 0.5
Late (67)	1.0 ± 0.4*	3.3 ± 1	207 ± 118*	77 ± 29	3.5 ± 0.8*

*p < 0.001 v. early

WEDNESDAY POSTER